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Issues in Osteoarthritis Care: Concepts and Controversies CME

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Goal

The goal of this activity is to explore the debate over osteoarthritis as a chronic inflammatory condition or a pain syndrome.

Learning Objectives

"Issues in Osteoarthritis Care: Concepts and Controversies" is intended for physicians and nurses*. Upon completion of this self-study activity, participants will be able to:

1. Compare the etiology and pathogenesis of osteoarthritis as a pain syndrome or chronic inflammatory condition.

2. Explore new and old treatment options for patients with osteoarthritis.
3. Identify nonpharmacologic management strategies for osteoarthritis patients.

Eligibility for Credit

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Instructions for Credit

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1. Register for continuing education credit by completing the "registration" process.
2. Read the learning objectives.
3. Read the article text and tables, and review figures.
4. Read, complete, and submit answers to the post test questions and evaluation questions. Participants must receive a test score of at least 70%, and respond to all evaluation questions to receive a certificate by mail. Certificates will be mailed to all eligible participants within 4-6 weeks after passing the post test and submitting the activity evaluation.

*Note: In most states, nurses are allowed to use CME accredited activities as evidence of having met requirements for continuing education participation in respect to license renewal.

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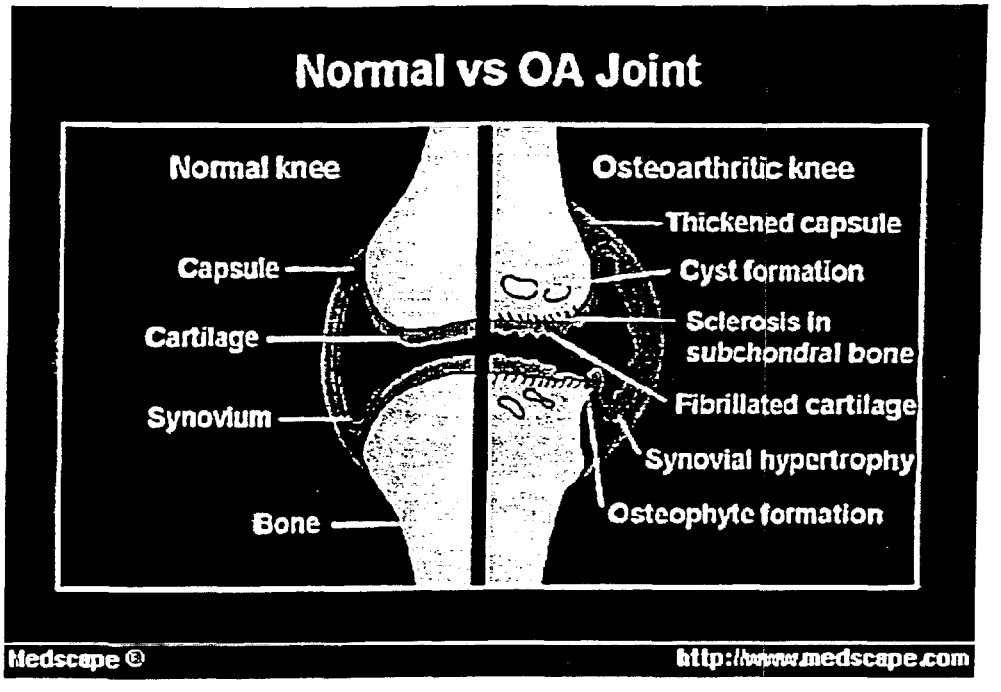
Introduction

One in six Americans has arthritis today, and an estimated one in five American citizens will have some form of the disease 20 years from now. Osteoarthritis (OA), the degenerative and more prevalent form of the disease, is characterized by articular cartilage degeneration and has long been a management challenge for both patient and physician. Relief of pain and functional disability is the main aim of OA therapy.[1] Treatment strategies can be broadly divided into nonpharmacologic and pharmacologic modalities.[2]



For many years, OA treatment options were limited, with physicians generally able to prescribe little more than standard analgesics or traditional anti-inflammatory agents.[3] A recent explosion in available new pharmacologic treatments has brought new hope for OA patients, offering therapeutic options not even dreamed of a decade ago.

But along with these advances has come a debate over whether OA treatment should focus on symptoms or on the underlying cause of the disease. This ongoing clinical controversy is rooted in the issue of whether OA is considered to be an inflammatory disorder or pain syndrome. Of import in this debate is the increase in research into new treatments for this condition, and a treatment paradigm that has been shifting in recent years. The Guidelines Committee for the American College of Rheumatology (ACR) published new OA treatment protocols as recently as 1995,[4,5] but rapid advances in therapy during the last 4 years have led to revision of these guidelines that will be published soon.

Until recently, physicians generally prescribed simple analgesic agents, such as acetaminophen, or one of the traditional nonsteroidal anti-inflammatory drugs (NSAIDs). Newer treatments that are equally efficacious, but with fewer side effects than established therapies, have now evolved, leading to a reassessment of the therapeutic schema for OA management.

Based on the presenting symptoms and the physician's judgment, simple analgesics and NSAIDs are primary considerations in first-line treatment of OA. Mild to moderate pain frequently responds to acetaminophen, while patients with more severe pain frequently respond better to NSAIDs, particularly when inflammation presents in the form of joint swelling, tenderness, and stiffness. Intra-articular corticosteroids are also used intermittently.

More recently, studies have shown intra-articular injections of hyaluronans to be efficacious with a high margin of safety in treatment of knee OA.[6] A relatively new class of drugs, the selective cyclooxygenase, COX-2 inhibitors, can provide anti-inflammatory and analgesic benefits with fewer gastrointestinal side effects than occur with traditional NSAIDs.[7-9] Accordingly, the availability of new agents and medications has led to a reassessment of therapeutic approaches in OA, although the COX-2 inhibitors have not yet been approved for use in many European countries.

A symposium at the recent ACR annual meeting addressed these management questions in a debate format. Members of the audience were able to participate in discussions by using keypads at their seats to answer the questions being posed by two leading authorities. Roy Altman, MD, of the University of Miami (Fla) School of Medicine, presented the argument that OA is primarily a chronic inflammatory condition, while Michael Doherty, MD, from the University of Nottingham (England) School of Medicine, presented the case that OA is primarily a pain syndrome.

Prior to the "Issues in Arthritis Care" debate, audience members were asked whether they considered osteoarthritis to be primarily a chronic inflammatory condition or primarily a pain syndrome. You are invited to offer your own opinion and compare it with those expressed by the audience, the demographics of which were as follows:

- **Practice location**
  - North America: 25%
  - South/Central America: 14%
  - Europe: 49%
  - Asia: 12%

- **Nature of practice**
  - University- or hospital-based: 60%
  - Private practice: 16.3%
  - Government- or state-employed: 6.7%
  - Multispecialty group: 4%
  - HMOs: 1.2%

With which of the following propositions do you agree?

A. Osteoarthritis is primarily a chronic inflammatory condition.	58.2 % (n=228)
B. Osteoarthritis is primarily a pain syndrome.	41.8 % (n=164)

The Inflammation Hypothesis

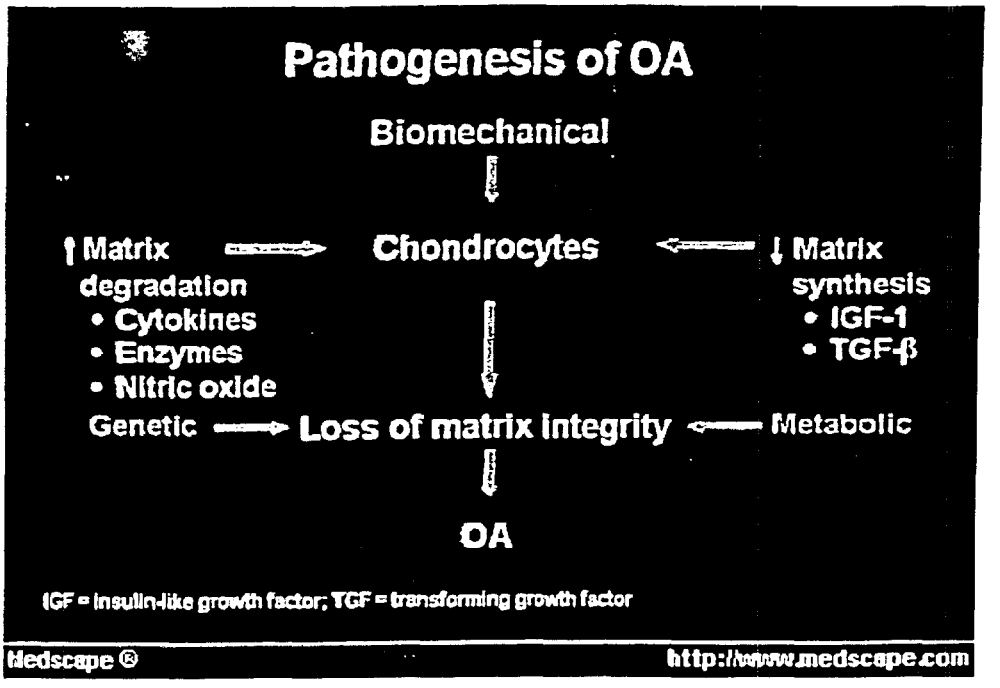
Dr. Altman argued that OA is primarily a chronic inflammatory condition. Although OA is generally not considered inflammatory because redness, heat, and other cardinal signs of inflammation are not always seen, pain and loss of function due to inflammation are present in most patients. The disease is only symptomatic in about half the afflicted patients.[10]

Evidence presented in favor of the inflammation hypothesis included increased uptake of technetium-99 label in the subchondral bone and synovium in OA joints in early-phase and late-phase bone scan and "angry" synovium characterized by edema and increased vascularity noted on arthroscopic evaluation.[11] C-reactive protein in serum is abnormally high and correlates with functional disability and joint tenderness. Although white blood cell count in normal synovial fluid is less than 100/mL on average, cellular response rises to about 800/mL or more in OA. This is a small increase compared with severe

inflammatory conditions, such as rheumatoid arthritis, in which the magnitude of the increase generally rises to thousands of cells per milliliter.

Crystals present in synovial fluid (calcium pyrophosphate and basic calcium phosphate) can activate the prostaglandin pathway leading to inflammation and cartilage damage. Synovial biopsy specimens on OA patients frequently show inflammatory synovitis severe enough in some cases that it cannot be distinguished from rheumatoid arthritis.

At the cellular level, interleukin-1, a potent cytokine, is increased in amount, leading to the release of inflammatory mediators and matrix metalloproteinases. In addition to its action in inhibiting proteoglycan synthesis, prostaglandins are increased in OA cartilage and synovium. Presentations at a recent meeting of the Osteoarthritis Research Society International emphasized the role of nitric oxide in OA, with some 28 papers related to nitric oxide and inflammation.[12] Additional papers focused on T-cell mediated inflammatory pathways.[13] Further evidence for the "inflammation hypothesis "relates to neurogenic transmission of inflammatory mediators inducing cartilage damage distant from the local site of inflammation.



A recent meta-analysis reported only one study failing to show nonsteroidals as less effective than acetaminophen, an analgesic agent, in the treatment of OA.[14] Another study compared levels of plasminogen activators and inhibitors in synovial fluid of knees from normal, OA, and rheumatoid arthritis patients; the study pointed out that the presence of these inflammatory compounds was abnormal not only in rheumatoid arthritis but also to some degree in OA.[15]

Recent studies comparing a combination of diclofenac and misoprostol with acetaminophen demonstrated statistically significant better responses to pain and relative function indexes for the diclofenac/misoprostol combination. Short-term studies reported similar superiority of ibuprofen over acetaminophen.

In summary, Dr. Altman affirmed that the presence of inflammation in OA both directly and indirectly, by noting a vast array of inflammatory indicators ranging from cytokines to growth factors that present themselves clinically, radiographically, and histologically.

The Pain Hypothesis

Dr. Doherty presented the case that OA is primarily a pain syndrome. He began by arguing that OA is

primarily a repair process by which a variety of insults trigger the need to restore, with all joint tissues (bone, cartilage, and synovium) involved in the response.

The triggering factors in an individual patient can often be identified; they are predominantly traumatic or mechanical, including rupture of the anterior cruciate ligament, meniscal derangement, or repetitive joint overusage in recreational activities. A truly inflammatory primary event may trigger subsequent OA, but that is unusual.

Dr. Doherty agreed with Dr. Altman in terms of the pathophysiologic process, citing elements in the OA tissue that are different from normal that represent inflammation. However, he argued that this was an integral part of the repair process. Although there are increased levels of cytokines and other catabolic mediators,[16] he proposed that these findings were secondary to the OA process. For example, the breakdown of cartilage results in the release of matrix components into the synovial cavity, leading to secondary synovitis, which in turn causes further cartilage breakdown due to synovial inflammation.

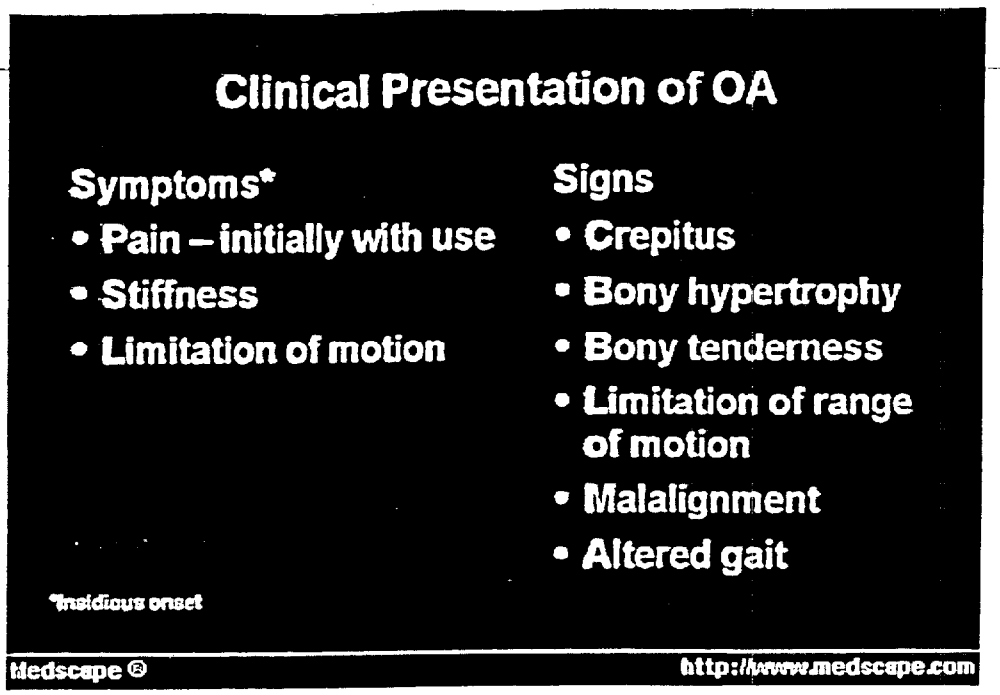
In many cases, the joint compensates for the insult in a slow repair process that is often efficient when done well. As a result, joints that are affected by OA have no symptoms in most patients. Thus, Dr. Doherty argued, OA itself does not necessarily produce pain and disability.

There is a relative minority of individuals in whom, for some unknown reason, the defense process does not work. Hyperplasia and increased inflammation occur in the cartilage, bone, and capsule with a modest degree in the synovium. But even more striking, unlike in inflammatory diseases, such as rheumatoid arthritis, is the thickening of the capsule, resulting from a loss of cartilage accompanied by increased subchondral bone.

In most true inflammatory conditions, however, there is a more generalized cartilage loss. A striking feature of OA is the exuberant hypertrophic new bone formation, osteophytes, present in most OA joints. This is an unusual feature in conditions, such as rheumatoid arthritis, that are primarily inflammatory. Dr. Doherty defined OA as "a joint that is damaged and is trying to repair itself which it may do quite well, rather than an atrophic primarily inflammatory problem."

Few people with OA have prominent isotope bone scan abnormalities in the early vascular study phase. The characteristic abnormality seen later is not due to inflammation of the bone, but rather to bone remodeling; apatite crystals of bone are taking up the nuclide. Accordingly, increased uptake does not necessarily represent inflammation.

In most patients with OA, physicians cannot define the reason, location, or mechanism for pain. In many cases, symptoms are often periarticular, and secondary bursitis and enthesopathy are common causes of discomfort in biomechanically altered OA joints. Intra-osseous hypertension, subchondral fractures, and other bone-related causes of the pain exist, and in some cases there may be early morning stiffness and effusion, but this is usually modest.



It is striking that when examining the triad at large (pain, disability, and structural change), often there is not a good symmetry among pain, disability and rapid pathophysiologic changes of OA. Reduced muscle strength, anxiety, and depression often correlate better with pain and disability than do radiographic and other structural changes. Therefore, it is not so much the rate or degree of radiographic severity or the amount of cytokines one may measure in synovial fluid that relates to these two; rather, they may be related to other features that we can more easily modify, Dr. Doherty argued.

In terms of treatment, Dr. Doherty noted that when considering large-joint OA, there is good evidence that long-term management strategies, such as muscle strengthening exercise, aerobic exercise, education, losing weight if the patient is obese, and lifestyle modifications may be significantly efficacious. These treatments are relatively effective and should form a central part of the management plan.

With respect to pharmacologic agents, Dr. Doherty reported that NSAIDs in particular have a poor track record. Although they are useful in many patients, their efficacy is low. Furthermore, NSAIDs have a variety of biological actions apart from blocking prostaglandin synthesis. They may hasten further damage to the joints and have long-term detrimental effects to cartilage and bone. In terms of efficacy, some patients clearly prefer NSAIDs to acetaminophen, for example. But clear superiority is hard to demonstrate, particularly in between-group comparison study designs. And the effect size of acetaminophen, which is simple and safe, is not often dramatic.

Furthermore, Dr. Doherty explained that it is often relatively easy to stop NSAIDs in OA patients without a substantial increase in symptoms. Several studies have confirmed that additional pain management strategies are not necessary when the NSAIDs are stopped. Another striking feature is that even if there is overt clinical evidence of inflammation it is often not a good predictor of the response to drug treatment, including NSAIDs, intra-articular steroid injections. Even though there may be inflammation in joints, it does not affect the way the patient's pain is controlled through pharmacologic therapy.

In summary, Dr. Doherty argued that multiple factors can trigger the process of repair in OA but that primary inflammation is a rare trigger. Most afflicted joints in OA are painless. Pain is an integral part of the repair process, and even though it is also part of inflammation, it is usually secondary.

Thus, Dr. Doherty said, physicians do not treat OA per se but rather treat people who have pain and disability. Associations of pain and structural OA differ, a positive point with regard to treatment, he

noted. Pain can be treated irrespective of the amount of OA activity or damage present. Furthermore, clinical assessments of inflammation should not guide the way therapies are prescribed.

The cornerstone of therapy, according to Dr. Doherty, is a nonharmful approach, important in the long-term management required in OA patients. Although useful analgesics and NSAIDs can be prescribed intermittently during the course of this disease, these agents should be added to treatment when patients are having exacerbations of their discomfort. First choice relates to nonpharmacologic approaches -- exercise, weight reduction, and avoidance of joint overuse.

Rebuttals

The speakers were given the opportunity to address each other's argument. Dr. Altman noted that inflammation is an essential part of the repair process. For example, an inflammatory response is required for blood clot removal in bone before fracture repair can proceed. Dr. Altman agreed that inflammation (as well as the pain) in OA is moderately cyclical so physicians do not necessarily need to treat people continuously for long periods of time.

Altman agreed that therapy could be discontinued when pain allowed but noted that evidence claiming NSAIDs as poor therapy for OA was limited to one study quoted by Dr. Doherty on indomethacin. Altman pointed out that there is no silver bullet for repairing the cartilage of the joint as a whole, and therefore, therapy is aimed at multifactorial elements that cause the symptoms. Altman stressed that analgesics and anti-inflammatory agents often need to be used in the treatment of OA, and physical measures should be used, but only as part of the therapeutic program.

Dr. Doherty replied that he was not disputing the inflammatory component of OA, but that joint inflammation does not necessarily cause pain. He asked if inflammation should be treated or should the physician just focus on the pain per se. As he noted earlier, structural changes and inflammation may have no relationship to the severity of pain or disability. Doherty asserted that physicians should monitor their patients, focusing purely on the pain, using the safest, simplest, and most effective therapy available. Although NSAIDs may be used, they are not needed in most patients.

During the rebuttal discussion, it was noted that the original ACR guidelines recommended use of acetaminophen as first-line therapy in all patients with OA; however, this is being reevaluated in light of the availability of the safer NSAIDs, the COX-2 inhibitors. Although perhaps 20% to 30% of patients have satisfactory responses to acetaminophen, more patients do well on NSAIDs. Deciding which therapy regimen to use depends on the patient's clinical picture and physician judgment, balancing the need for efficacy and safety.

Audience Questions

A number of questions and comments from the audience were subsequently addressed. It was suggested that although some studies reported a negative influence on joint structure, chronic suppression of synovitis might actually be chondroprotective. Dr. Doherty commented that whether NSAIDs had a positive or negative effect on OA structures was a crucial question. In Europe, as new drugs are increasingly being used for OA, there is a need to provide evidence of musculoskeletal safety in terms of long-term radiographic changes.

Dr. Doherty pointed out that the only human data currently available do not suggest that NSAIDs are protective. These agents may be disease modifying, but in a negative fashion, actually exacerbating the disease process. Bone changes related to the new COX-2 inhibitors have not yet been reported. The available evidence supports the theory that NSAIDs stop new heterotopic bone formation.

Dr. Altman replied that there was limited information about the negative impact on joint structure by NSAIDs. He pointed out that there is one NSAID used in Europe that appeared to be protective in animal models of OA and had no deleterious effect with respect to joint breakdown in studies in humans.

A question was posed as to what degree economics dictate which treatments are used by physicians, compared with what the physician thinks is best for the patient. Dr. Doherty replied that with the constraints of modern health care, a physician must make appropriate decisions, which often can be difficult. For example, decisions relating to the use of extremely expensive disease-modifying drugs in people with rheumatoid arthritis and systemic lupus erythematosus can be particularly difficult.

Dr. Altman concurred that the cost of the medication is important in the therapy for patients; managed care in particular has forced practicing physicians to pay more attention to the cost of agents. At the Veterans Affairs Hospital in Miami, Fla, the cost of agents are written on the pharmacy sheets, so that clinicians are reminded to consider cost when choosing therapies.

A question was posed as to whether there was a nephrotoxic risk in using NSAIDs like acetaminophen together. As noted in the past with acetaminophen and phenacetin, the answer was risk of renal toxicity was not likely.

An audience member asked, if COX-2 inhibitors are safer, why aren't they routinely recommended? He also wanted to know if it would be considered malpractice if a complication occurred due to continued the use of traditional NSAIDs. The speakers pointed out that at present a number of health maintenance organizations do not allow the use of COX -2 inhibitors until patients either fail traditional NSAIDs or have a complication with them.

Symptoms are phasic in OA and using anti-inflammatory agents and acetaminophen at different phases of disease was appropriate. One audience participant noted that not all patients were going to need a lifetime of therapy, whether it be analgesic or anti-inflammatory.

A question was entertained as to whether patients and physicians would use acetaminophen more often if it came in a once-daily dosage. The point was made that multiple dosing may have an effect on patient satisfaction. In Dr. Altman's experience, most patients prefer to control their pain medications, using it as pain comes and goes during the day. On the other hand, it was noted that in situations in which medications must be taken three or four times a day, some patients might be less compliant.

So What Have We Learned?

In summarizing the ongoing debate over how best to treat OA, it's important to consider not only differing evidence and opinion but to keep in perspective current recommendations for management of this condition.

The marked changes that have taken place in the last several decades with regard to OA management are clear from the increasing number of modalities available for effective treatment. Rheumatology therapeutics has become an extremely exciting area, with more advances in the past decade than have probably been seen during the previous 30 or 40 years subsequent to the discovery of cortisone.

Still, the question remains: just what are physicians treating in OA? Treatment involves focusing on pain as the primary symptom, but the focus also includes treating swelling and inflammation. The cause of pain is multifactorial, so therapy needs to address specific pathophysiologic components related to disease mechanisms. Pain in OA is related both to inflammatory and noninflammatory causes, including synovitis, release of cytokines, muscle spasm, periostial elevation in the form of osteophytes, subchondral fractures, and inter-osseous hypertension.

Should treatment, therefore, focus on pain or the causes of pain? Should physicians treat the pain with simple analgesics, or should they treat inflammation and pain with NSAIDs. Several examples were cited to illustrate the point of these rhetorical questions. Angina is not solely treated with opioids, rather the vasospasm that causes the pain is treated with nitroglycerine-related drugs. Most clinicians would not agree to using opioids as the only treatment for a patient with ischemic cardiac symptoms. Similarly, with respect to migraine headaches, the pain is often controlled with opioids, but now there is a proven benefit in treating the altered vascular reactivity, the cause of the pain.

In the Guidelines for Osteoarthritis[4,5] (Figure 1) published under the auspices of the ACR in *Arthritis and Rheumatism* in 1995, it was recommended that the treatment of OA include a basic umbrella program. The recommendation included patient education, weight loss, and physical therapy, followed by use of acetaminophen up to full doses (1 g, 4 times a day), low-dose over-the-counter NSAIDs, and full-dose NSAIDs, with the use of intra-articular corticosteroids judiciously spaced.

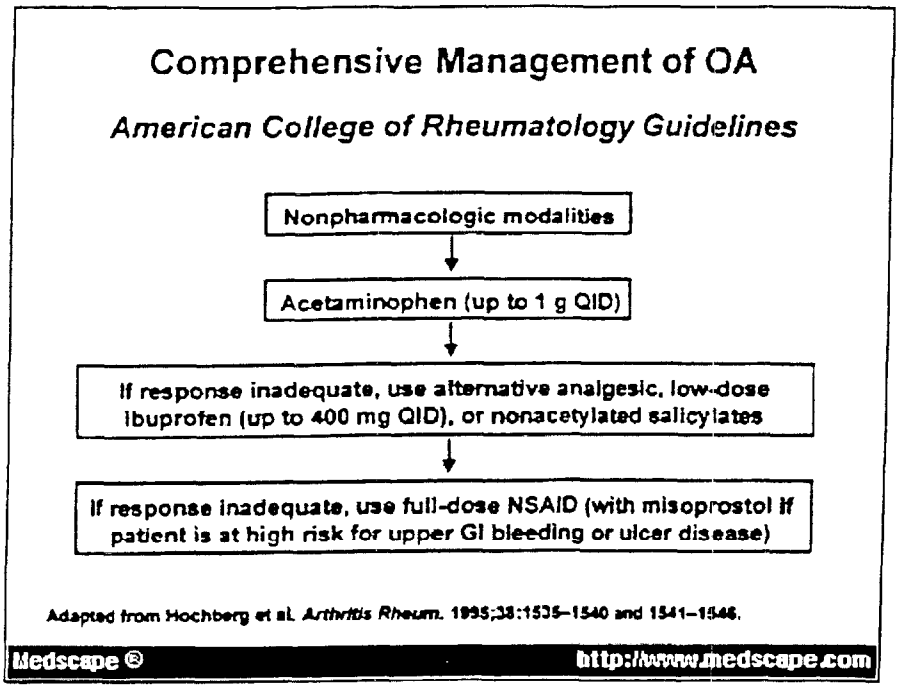


Figure 1. Schema of ACR guidelines for OA of the knee.[5]

Perhaps it is time to suggest a new therapeutic schema (Figure 2), which stresses that the agent being used in initiating OA therapy in a given patient depends on that patient and his or her individual needs. In some patients who have mild to moderate pain, acetaminophen will be effective. For many patients, however, presenting with moderate or severe pain, particularly with inflammation in the form of stiffness, joint tenderness or effusion, COX-2-specific NSAIDs, or traditional NSAIDs with gastroprotective agents are likely to provide higher degrees of benefit. Although COX-2-specific NSAIDs and traditional NSAIDs have similar efficacy, the COX-2-specific NSAIDs have a higher safety profile. A physician can start with one agent and go on to the other -- moving from anti-inflammatory agents to analgesics or vice versa, depending on patient symptomatology. Other analgesics, such as tramadol, propoxyphene, or carefully used opioids, should also be considered. Intra-articular hyaluronans and intra-articular corticosteroids could be used appropriately at different intervals.

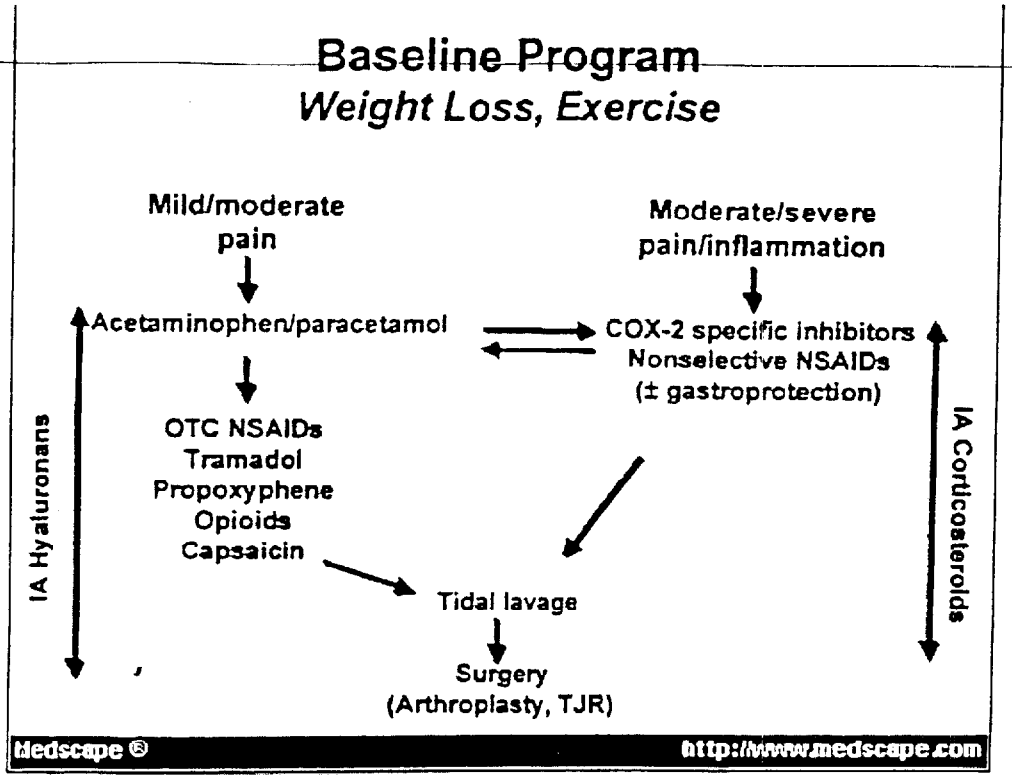


Figure 2. Proposed new schema for treatment of knee OA.

Voltaire said that "doctors pour drugs of which they know little to cure diseases of which they know less into human beings of whom they know nothing." Times have changed. Physicians now know a great deal about the drugs they use today, about the people whom they are treating, and about the diseases that they and their patients face. As a result, at least in the case of OA, physicians and patients both can look forward to more treatment options and less frustration.

During the "Issues in Arthritis Care" symposium, audience members were asked a number of questions about their use of various therapeutic agents in the treatment of osteoarthritis, based on different clinical features that patients might present. Answers were recorded by means of keypads at respondents' seats. A list of these questions follows. You are invited to take the same survey as the symposium audience and compare your answers with theirs.

Question 1: A 65-year-old woman presents with a history of chronic OA of the right knee with no synovial swelling or tenderness. Your initial treatment would consist of which of the following:

A. Acetaminophen/paracetamol	61.9% (276 people)
B. Traditional NSAID	10.5% (47)
C. COX-2-specific inhibitor	24% (107)
D. Intra-articular hyaluronan	1.8% (8)
E. Intra-articular corticosteroid	1.8% (8)

Question 2: The same patient presents with OA of the knee associated with morning stiffness, mild effusion, and tenderness to palpation. Your initial therapy for this patient would be:

A. Acetaminophen/paracetamol	4.7 % (21)
B. Traditional NSAID	27.6% (123)
C. COX-2-specific inhibitor	48.5 % (216)
D. Intra-articular hyaluronan	0.9 % (4)
E. Intra-articular corticosteroid	18.2 % (81)

Question 3: A 40-year-old male presents with secondary OA subsequent to a tear of the anterior cruciate ligament some 10 years previously. Your initial therapy would be which of the following:

A. Traditional NSAID	24 % (95)
B. Traditional NSAID plus a proton-pump inhibitor	7.6% (30)
C. Acetaminophen/paracetamol	20.7% (82)
D. COX-2-specific inhibitor	32.8% (130)
E. Intra-articular hyaluronan	14.9 % (59)

Question 4: In your experience, do patients treated with NSAIDs do better symptomatically than patients receiving full doses of acetaminophen/paracetamol?

A. Yes	74.9 % (281)
B. No	13.3% (50)
C. Undecided	11.7% (44)

Question 5: Would you say that the majority of patients with OA do well on a program of nonpharmacologic approaches (weight reduction, exercises, avoidance of overuse) plus acetaminophen/paracetamol in full doses?

A. Yes	52.2 % (201)
B. No	42.1 % (162)
C. Undecided	5.7 % (22)

Question 6: COX-2-specific inhibitors are sufficiently advantageous with respect to safety to warrant their preferential use as the primary NSAID in beginning anti-inflammatory therapy in OA patients:

A. Yes	67.9 % (285)
B. No	20.7 % (87)
C. Undecided	11.4 % (48)

Question 7: Although COX-2-specific inhibitors appear to be safer than traditional NSAIDs, would you continue to use non-COX-2-specific NSAIDs in patients with uncomplicated OA?

A. Yes	57.4 % (224)
B. No	36.2 % (141)
C. Undecided	6.4 % (25)

Question 8: Suppression of inflammation of OA is hypothetically advantageous since it may result in slowing down the degenerative process.

A. Agree	66.1 % (226)
B. Disagree	18.7 % (64)
C. Undecided	15.2 % (52)

Question 9: A 72-year-old otherwise healthy man presents with acute back symptoms for which you anticipate therapy for 2 weeks. Your choice of therapy for this short-term treatment would be:

A. A COX-2-specific inhibitor	48.5 % (183)
B. A traditional NSAID	20.7 % (78)
C. A traditional NSAID plus misoprostol	9.0 % (34)
D. Acetaminophen/paracetamol	21.8 % (82)

Question 10: If this patient had a history of ulcer disease, your choice of therapy for this short-term treatment would be:

A. A COX-2-specific inhibitor	64.6 % (226)
B. A traditional NSAID	0.6 % (2)
C. A traditional NSAID plus misoprostol	9.4 % (33)
D. Acetaminophen	25.4 % (89)

Question 11: If this patient with a history of ulcer disease had chronic OA of the knee requiring long-term therapy, your choice of treatment would be:

A. A COX-2 specific inhibitor	63.9 % (242)
B. A traditional NSAID	0.8 % (3)
C. A traditional NSAID plus misoprostol	7.1 % (27)
D. Acetaminophen	28. 2 % (107)

Question 12: A 69-year-old otherwise healthy woman requires long-term therapy for OA of both knees and the spine. A 1-g dose of acetaminophen/paracetamol 4 times a day has not adequately controlled the symptoms. You would select which of the following therapies:

A. Traditional NSAID plus misoprostol	10.8% (35)
B. COX-2-specific inhibitor	70.4 % (228)
C. Traditional NSAID alone	6.8 % (22)
D. Acetaminophen with codeine/paracetamol three times a day	12 % (39)

After the debate, the audience was again asked whether they considered osteoarthritis to be primarily a chronic inflammatory condition or primarily a pain syndrome. Now that you've heard the evidence and opinions and taken the survey, you are invited to offer your own opinion, regardless of whether it has changed, and compare it with those expressed by the audience.

With which of the following propositions do you agree?

A. Osteoarthritis is primarily a chronic inflammatory condition.	57.1 % (n=137)
B. Osteoarthritis is primarily a pain syndrome.	42.9 % (n=103)

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Suggested Reading

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Issues in Osteoarthritis Care: Concepts and Controversies CME

Post-Test

Click on the appropriate response. A test score of 70% or greater is required for accreditation.

1. All of the following are observed in osteoarthritis (OA) except:

- A. Increased uptake of technetium-99 label in the subchondral bone and synovium in OA joints.
- B. Edema and increased vascularity noted on arthroscopic evaluation of the synovium.
- C. Abnormally low C-reactive protein in serum.
- D. Synovial fluid cellular response rarely exceeds a white blood cell count of 800/mL.

---

2. In many inflammatory conditions there is a generalized cartilage loss. A striking feature of OA is:

- A. Exuberant hypertrophic new bone formation called osteophytes present in the majority of affected joints.
- B. Extensive, generalized loss of subchondral bone.
- C. Thinning of the capsule resulting from a decrease in subchondral bone.
- D. Severe soft-tissue swelling.

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3. Common triggering factors in patients with OA can often be identified and include all of the following except:

- A. Rupture of the anterior cruciate ligament.
- B. Presence of a septic joint.
- C. Meniscal derangement.
- D. Repetitive joint overuse in recreational activities.

---

4. Although COX-2-specific NSAIDs and traditional NSAIDs have similar efficacy, the COX-2-specific NSAIDs have:

- A. more side effects.
- B. a longer half-life.
- C. a higher safety profile.
- D. none of the above.

---

5. There is general agreement that standard initial therapy in patients with chronic OA of 1 knee with no synovial swelling or tenderness should be:

- A.Traditional NSAIDs.
- B. Acetaminophen/paracetamol.
- C. COX-2 inhibitor.
- D. Intra-articular injection.
- E. Individualized depending on the presence or absence of inflammation.

Issues in Osteoarthritis Care: Concepts and Controversies CME

Evaluation

Scale: 5 = Excellent 4 = Good 3 = Satisfactory 2 = Fair 1 = Poor

1. How would you rate how well you can achieve the following learning objectives?

a. Compare the etiology and pathogenesis of osteoarthritis as a pain syndrome or chronic inflammatory condition.

O5 O4 O3 O2 O1

b. Explore new and old treatment options for patients with osteoarthritis.

O5 O4 O3 O2 O1

c. Identify nonpharmacologic management strategies for osteoarthritis patients

O5 O4 O3 O2 O1

2. How would you rate the relevance of activity content to the objectives?

O5 O4 O3 O2 O1

3. How would you rate the faculty's effectiveness (clarity and organization) in presenting the material?

O5 O4 O3 O2 O1

4. How would you rate the content?

O a. Will definitely change the way you practice
O b. Challenged you to think about the topics
O c. Applicable to your practice; a good review
O d. Of limited use in your practice
O e. Not applicable to your practice

5. How well did this activity meet the goal of providing state-of-the-art treatment protocols and clinical management strategies?

O5 O4 O3 O2 O1

6. Were your personal objectives for taking this activity met?

O5 O4 O3 O2 O1

7. How long did this session actually take you to complete?

O a. .25 - .50 hrs
O b. .50 - 1.0 hrs
O c. 1.0 - 1.5 hrs
O d. 1.5 - 2.0 hrs
O e. More than 2.0 hrs

8. What other continuing education topics would be of value to you? Please offer any additional comments.

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